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Application of neurally adjusted ventilatory assist in neonates☆

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SUMMARY

Neurally adjusted ventilatory assist (NAVA) uses the electrical activity of the diaphragm (Edi) as a neural trigger to synchronize mechanical ventilatory breaths with the patient's neural respiratory drive. Using this signal enables the ventilator to proportionally support the patient's instantaneous drive on a breath-by-breath basis. Synchrony can be achieved even in the presence of significant air leaks, which make this an attractive choice for invasive and non-invasive ventilation of the neonate. This paper describes the Edi signal, neuroventilatory coupling, and patient–ventilator synchrony including the functional concept of NAVA. Safety features, NAVA terminology, and clinical application of NAVA to unload respiratory musculature are presented. The use of the Edi signal as a respiratory vital sign for conventional ventilation is discussed. The results of animal and adult studies are briefly summarized and detailed descriptions of all NAVA-related research in pediatric and neonatal patients are provided. Further studies are needed to determine whether NAVA will have significant impact on the overall outcomes of neonates.

1. Introduction

Diaphragmatic electromyography (EMG) was first used by Petit in 1959 to evaluate respiratory muscle function [1]. Since then, diaphragmatic EMG has been utilized by a variety of investigators, between 1983 and 1994, to study diaphragmatic activity, sleep state and response to CO_2 in infants [2–5]. In 1987 Daubenspeck et al. described a new technique to evaluate the diaphragmatic EMG using an array of seven sequential electrode pairs on an esophageal catheter [6]. In the 1990s Sinderby and Beck expanded this concept with the introduction of embedded electrodes in a nasogastric tube that detected a reliable diaphragmatic EMG signal. This signal reflects the patient's neural respiratory drive in real time, and minimizes artifacts and noise [7–9]. This new, minimally invasive, bedside technology has been integrated into a commercially available mechanical ventilator (Servo-I; Maquet, Solna, Sweden) that converts this electrical activity into a proportionally assisted and synchronized breath known as neurally adjusted ventilatory assist (NAVA) [9–11].

2. Electrical activity of the diaphragm (Edi)

The diaphragmatic EMG is also referred to as the electrical activity of the diaphragm (Edi). The magnitude of this diaphragmatic activation (and hence the Edi signal) is controlled by adjusting the nerve fiber recruitment (the number of nerves that are sending the stimulus) and the rate coding (stimulation frequency). A specialized nasogastric tube, containing an array of eight bipolar electrodes (sensors are placed above the feeding holes), is positioned in the lower esophagus at the level of the crural diaphragm to measure the Edi signal. The position of the diaphragm is determined along the electrode array [12] and the double subtraction technique is then applied to the electrode pairs close to the diaphragm [13]. Signals from each electrode pair are differentially amplified, digitized, and processed. The signal is filtered to remove electrical contamination from the heart, esophagus, and environment to give the highest possible signal-to-noise ratio [10]. Measuring the Edi in the esophagus with the double subtraction method removes potential contamination from changes in lung volume, body position, intra-abdominal pressure, postural and expiratory muscles, the subcutaneous layers, and applied positive end-expiratory pressure (PEEP) [7,8,10,14–16]. Signal integrity also does not seem to be influenced when receiving bolus versus continuous nasogastric feeds [17] or with milk influx during oral feeding [18]. Failure to detect an Edi signal may be from failure of the respiratory center to deliver a signal (e.g. apnea of prematurity, central hypoventilation syndrome, over-assist, hyperventilation, brain injury, sedation),

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anatomic reasons (diaphragmatic hernia), or peripheral abnormalities (e.g. phrenic nerve conduction failure, disease, or chemical paralysis of the neuromuscular junction or diaphragm) [10]. Edi has been shown to correlate with transdiaphragmatic pressure [7,19,20] and with the pressure generated by the respiratory muscles and inspiratory effort [21].

Initial placement of the specialized nasogastric tube (Fig. 1) is determined like any other nasogastric tube by using the measurement of nose–ear–xiphoid distance. The position can then be verified and adjusted using the retrocardiac electrocardiography (EKG), obtained from the electrodes on the catheter. The dedicated catheter-positioning screen located on the Servo-I (Fig. 2), displays these waveforms. Correct nasogastric catheter position is demonstrated by the largest p-waves and by QRS complexes being present in the upper leads and subsequently progressing to minimal or absent p-waves and QRS complexes in the lower leads. The Edi signal is superimposed on the retrocardiac EKG as a blue color and should be on the second and third lead but may periodically fluctuate to the upper and lower leads without loss of signal integrity.

3. Control of neuroventilatory coupling

During spontaneous breathing electrical excitation of the diaphragm occurs when the respiratory signal originating in the brainstem travels via the phrenic nerve to the diaphragm. The diaphragm then contracts, resulting in expansion of the chest muscles causing negative pressure in the chest so that air is drawn into the lung. The lung subsequently expands with changes in pulmonary pressures, flow, and volume (Fig. 3) [14,22]. A variety of biological sensors provides neural feedback and adjusts the respiratory drive with each spontaneous breath. This involves a very complex regulatory system including stretch receptors in the lung, the Hering–Breuer reflex, lung compliance changes, upper airway receptors, peripheral chemoreceptors in the carotid body, and central chemoreceptors located in the brain stem [23,24].

4. Patient–ventilator synchrony

Mechanical ventilation provides appropriate unloading of the respiratory muscles and maintains adequate gas exchange until the respiratory disease that is responsible for the patient's respiratory insufficiency has improved [10,25]. Diaphragmatic dysfunction and atrophy have been associated with short-term ventilation of $\leq$7 days [26–28], long-term ventilation of $>$12 days, and failure of normal pulmonary growth and maturation [28]. Ventilator management with partial support modalities that permit diaphragmatic effort or promote periods of spontaneous breathing have been shown to alleviate some of the ventilator-induced dysfunction (VIDD) [29]. The best current approaches to avoid VIDD and possibly ventilator-induced lung injury (VILI) include strategies to promote synchronous ventilation, minimize mechanically delivered tidal volumes, and avoid both controlled mechanical ventilation and the use of paralytic agents [29,30].

The goal of synchronous ventilation is to provide a mechanical breath that is synchronous for timing of inspiratory effort and assists proportionally to the patient's needs [10]. Cycling is controlled either by time or by flow, but the support level cannot be controlled proportionally to the patient's effort or demand breath-by-breath. Inappropriate choice of pressure support levels may lead to ventilatory over-assistance, resulting in suppression of the patient's respiratory drive, and may lead to wasted respiratory effort by the patient [31]. Proportional assist ventilation (PAV) offers an assist level (pressure or volume) proportional to patient-generated flow and volume, in addition to inspiratory triggering and cycling (controlled either by time or flow) [25]. All three types of ventilatory support [pressure support ventilation (PSV), pressure-controlled ventilation (PCV) or PAV] respond to the patient's mechanical respiratory effort but not neural (central) respiratory need or drive. The breaths are initiated only after the neuroventilatory cascade is complete (denoted as flow trigger in Fig. 3).

Asynchronous ventilation has the potential for adverse effects, which include the need for increased mean airway pressure, higher

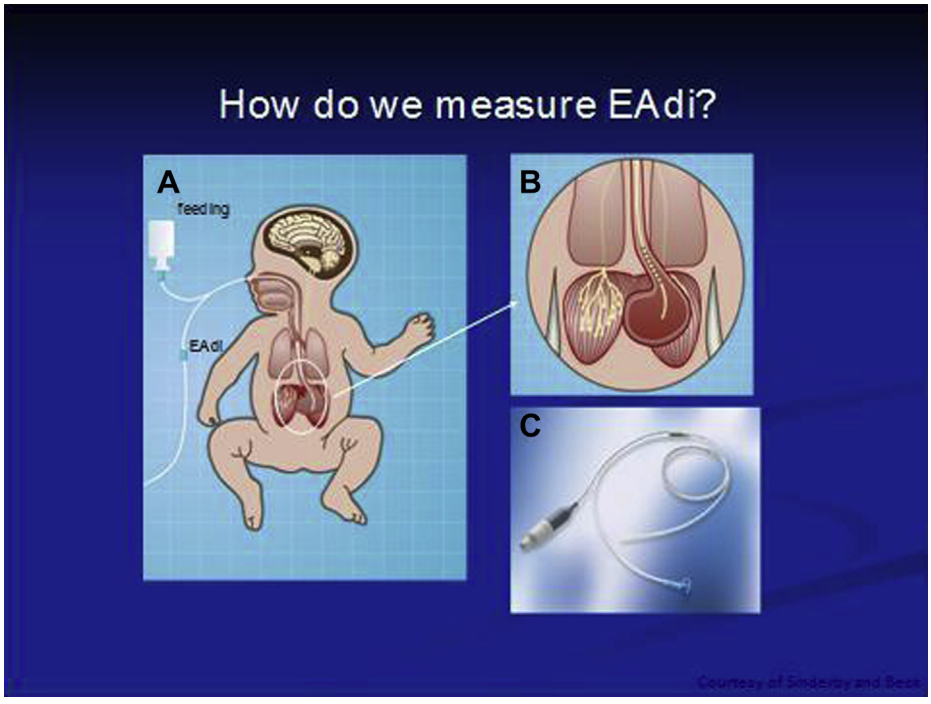

Fig. 1. Positioning of the specialized nasogastric tube with electrodes above and below the diaphragm. (Reprinted with permission from J. Beck and C.A. Sinderby, Toronto, Ontario.)